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VExUS score: optimizing its use in perioperative and critical care management

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Abstract

The Venous Excess Ultrasound (VExUS) score has been developed to grade venous congestion and predict acute kidney injury in cardiac surgical setting by combining inferior vena cava diameter with hepatic, portal, and renal venous Doppler patterns. While initial studies demonstrated associations with acute kidney injury, subsequent non cardiac setting ICU studies yielded contradictory results, prompting reevaluation of what VExUS actually measures. Analysis of VExUS components reveals the score reflects complex interactions between cardiac function, filling pressures, and volume status rather than pure volume assessment. Each component—IVC diameter, hepatic vein flow, portal vein flow, and renal vein flow—is differently influenced by cardiac function and volume status. Observational studies demonstrate that patients with highest VExUS scores often have cardiogenic shock despite low fluid balance, while those with significant volume excess may have low scores when cardiac function is preserved. This evolving understanding necessitates a correction in clinical practice, returning to the original physiological framework where VExUS reflects the interaction of venous return and cardiac function rather than simple volume overload parameter.

Keywords Congestion, Acute kidney injury, VExUS, Cardiogenic, Shock, Volemia, Sepsis

The Venous Excess Ultrasound (VExUS) score has emerged as a promising tool in perioperative and critical care medicine to improve our approach to fluid management and venous congestion assessment [1]. However, as evidence accumulates from literature, a more nuanced understanding of what VExUS actually measures is emerging.

The promise and the reality

When the VExUS was introduced, it seemed to offer an elegant solution to a complex problem: how to non-invasively assess venous congestion and predict organ dysfunction? This score was constructed by combining maximal inferior vena cava (IVC) diameter with assessment of venous Doppler patterns of the hepatic, portal, and renal veins. In the original study, several VExUS scores were described (A to E), but VExUS type C was the one that was associated with cardiac surgery acute kidney injury (AKI) [1]. VExUS type C is graded as follows; grade 0: no IVC dilatation regardless of other patterns, grade 1: IVC diameter over 2 cm with normal or mild patterns of abnormalities; grade 2 (mild congestion): IVC diameter over 2 cm with one severe abnormality in at least one pattern; and grade 3 (severe congestion): IVC diameter over 2 cm with severe abnormalities in multiple patterns. Since this first publication, many

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studies evaluating VExUS as an easy, non-invasive tool for assessing venous congestion and organ dysfunction have been published with contradictory findings [2, 3]. Given that many studies have failed to demonstrate a consistent association between VExUS scores and AKI in general ICU populations, the discrepancy between the cardiac setting (where associations are stronger) and ICU populations may suggest something different than originally intended.

Understanding what VExUS actually measures

To better understand what VExUS measures, the first step should be to understand what each component of VExUS measures and means. As previously described, construction of the VExUS score is based on the IVC maximal diameter, the hepatic venous flow, the portal venous flow, and the renal venous flow. Throughout the manuscript, when referring to cardiac function influencing VExUS components, we specifically discuss cardiac function as a whole that comprise: right ventricular (RV) diastolic and/or systolic function, RV afterload and RV–pulmonary arterial coupling, as well as left ventricular (LV) diastolic and/or systolic function, and ventriculo-arterial coupling. Thus, physician should check each component of the cardiac function.

Measuring IVC diameter represents the first step in grading the VExUS score. While multiple factors beyond volume status can influence IVC diameter, a dilated IVC can be a reflection of elevated right atrial pressure and/or RV dysfunction [4, 5]. Although a tremendous number of studies have evaluated the best method (formula, IVC diameter and collapsibility) to evaluate right atrial pressure from IVC measurement, the main finding is that IVC depends on too many factors to be used solely to evaluate right atrial pressure [6]. To illustrate this point, a wide IVC diameter can be observed in several situations, such as during positive-pressure ventilation (which increases intrathoracic pressure and reduces venous return), with vigorous spontaneous breathing efforts (causing dynamic diameter changes), in cirrhotic portal hypertension, or in patients with tricuspid regurgitation (leading to IVC dilation without necessarily indicating hypervolemia).

Once IVC diameter has been assessed, other venous ultrasound veins should be assessed. Close to the right atrium, the hepatic veins connect liver blood flow to the vena cava and venous return. The shape of the hepatic vein flow is influenced by changes in pressure in the right atrium. But these changes depend on a complex interaction between right atrial function, right ventricular function, and volume [7]. Given that the hepatic vein Doppler pattern (S/D ratio) is directly influenced by RV contraction and tricuspid valve movement, an abnormal (inverse S/D ratio) pattern primarily reflects RV function rather than hypervolemia per se [8, 9].

Additionally, portal vein pulsatility is the consequence of a complex interplay between cardiac function, splanchnic capacitance, and volume status [10]. Intrinsic hepatic disease can also alter portal vein waveform patterns independently of cardiac function. This may result from decreased hepatic compliance and/or portal hypertension, which can cause portal vein pulsatility. Interpretation of a high portal pulsatility index may therefore be challenging, as studies have shown that both cardiac function and volume status influence portal flow pulsatility [11–13].

Lastly, renal venous flow patterns, while influenced by volume, can be affected by cardiac function and local renal hemodynamics [14, 15]. Local renal hemodynamic factors may include renal interstitial edema, which increases intrarenal pressure and compresses venous outflow; reduced arterial and venous compliance due to sympathetic activation; decreased renal arterial inflow from hypoperfusion, which alters the arterial–venous pressure gradient; or elevated intra-abdominal pressure, which directly compresses the renal vasculature and increases external venous resistance. In addition to these local factors, cardiac function (both diastolic and systolic) can also influence renal venous flow in the setting of hypervolemia, through elevated right- and left-sided filling pressures—a mechanism improved with volume depletion induced by diuretics [15]. Subsequently, a study in the ICU confirmed that portal flow pulsatility and venous renal Doppler may be associated with an appropriate response to diuretic volume depletion [12]. The presence of abnormal venous renal patterns may indicate volume overload that would benefit from diuresis.

Based on these observations, physicians should understand that VExUS reflects the interaction between cardiac function, cardiac filling pressures, and volume status. In other words, VExUS reflects the interactions between venous return and cardiac function curves, determined by the stressed volume, the vascular compliance (both determining mean systemic filling pressure), and cardiac function (particularly RV function including contractility, afterload and RV to pulmonary artery coupling). An observational study focusing on type of congestion in ICU has observed that patients with the highest VExUS scores were those suffering from cardiogenic shock despite having negative to low positive fluid balance [16]. Also, patients with higher positive fluid balance had pulsatility of portal venous flow but given that right and left ventricular functions were within normal range, the VExUS score was low [16]. This observation is confirmed by physiologic studies demonstrating that portal flow becomes pulsatile with fluid expansion in healthy subjects for whom left and right ventricular functions are assumed to be normal [17]. Given this assumption, we can better understand several contradictory

observations. A notable example is the coexistence of high VExUS grades and preload dependence. Patients with high VExUS grades may still demonstrate fluid responsiveness, as VExUS reflects the inability of the cardiovascular system to accommodate existing volume at acceptable filling pressures rather than absolute hypervolemia [18]. Elevated filling pressures from impaired cardiac function can coexist with preserved Frank–Starling reserve, allowing an increase in cardiac output with additional preload. Accordingly, fluid balance does not necessarily correlate with VExUS grade, which primarily reflects the pressure consequences of intravascular volume relative to cardiac function [19]. In this sense, a medical strategy based on VExUS can better reduce cardiac-driven congestion without improving kidney injury resolution [20]. This is why, when evaluating cardiovascular factors associated with VExUS, the main determinants are primarily the left and right ventricular filling pressures (and their derivatives) [21]. Taking these factors in account explain why VExUS should not be used as the only trigger to treat cardiorenal syndrome, and why it failed to assess AKI in non-cardiac setting [2, 22]. VExUS confirm high probability of elevated ventricular filling pressure [21, 23]. Finally, a meta-analysis evaluating the association between the VExUS and AKI confirmed that such an association is mainly observed in cardiovascular settings [3].

Clinical implications: a paradigm shift

This evolving understanding necessitates a fundamental shift in how we should interpret the VExUS:

From volume assessment to cardiac evaluation: Rather than understanding the VExUS as a volume status indicator, we should recognize it primarily as a cardiac-driven congestion score that rely on cardiac function and volume status [16]. A high VExUS score should prompt comprehensive evaluation of biventricular cardiac function, including both systolic and diastolic parameters [23, 24].

Context-dependent interpretation: The clinical context becomes paramount. A patient with known right ventricular dysfunction may have an elevated VExUS score despite being euvolemic. Conversely, a patient with normal cardiac function might tolerate significant hypervolemia without manifesting high VExUS scores [16]. VExUS should never be interpreted in isolation. Integration with clinical assessment, hemodynamic parameters, tissue perfusion parameters and cardiac ultrasound assessment is essential.

Clinical integration with other parameters (Fig. 1) When the VExUS score is higher than grade 1, the main consideration is to understand whether the patient suf-

fers from volume-driven congestion, cardiac-driven congestion or both. To answer this question, given that each component of the VExUS score is associated with either volume or cardiac-related congestion, a balanced analysis can help identify the main underlying cause.

The first step is to analyze each component of cardiac function and assess portal flow pulsatility. High portal flow pulsatility combined with altered patterns of cardiac function suggests volume and cardiac-driven congestion. This represents a mixed pathophysiology where both hypervolemia and cardiac dysfunction contribute to the clinical presentation. The therapeutic approach must address both components, requiring a combination of diuretics for fluid removal along with inotropic and/or inodilator treatments for systolic cardiac function improvement and afterload reduction thus improving ventriculo-arterial coupling.

When cardiac function assessment reveals abnormalities but portal flow patterns remain normal, the congestion becomes primarily cardiac-driven. In this scenario, analyzing the hepatic vein flow can reveal abnormal patterns confirming the cardiac etiology. In this case, analyzing the renal venous flow pattern may confirm or refute the diagnosis. A normal pattern confirms the diagnosis whereas an abnormal pattern may suggest a part of volume-driven congestion. When faced with this clinical scenario, volume removal alone (abnormal venous renal pattern) may be insufficient or potentially harmful (normal venous renal pattern). The therapeutic focus shifts to cardiac function optimization.

Conversely, when cardiac function is normal, an abnormal portal pulsatility pattern may indicate volume-driven congestion that may be treated with fluid removal. In this case, analyzing the renal venous flow pattern may confirm the diagnosis with an abnormal venous renal pattern [12]. An often-overlooked clinical consideration is that patients with volume-driven congestion can paradoxically still be preload dependent. This means that despite a high VExUS score, these patients may still increase cardiac output with fluid administration, making fluid removal potentially inappropriate without careful tissue perfusion and hemodynamic assessments [25].

Conclusions

The VExUS score represents an important advance in bedside ultrasound assessment, but its true value may lie in a different domain than originally envisioned. By recognizing VExUS as a tool reflecting the interaction between venous return and cardiac function rather than a pure volume-driven congestion score, we can use it more appropriately to guide patient care.

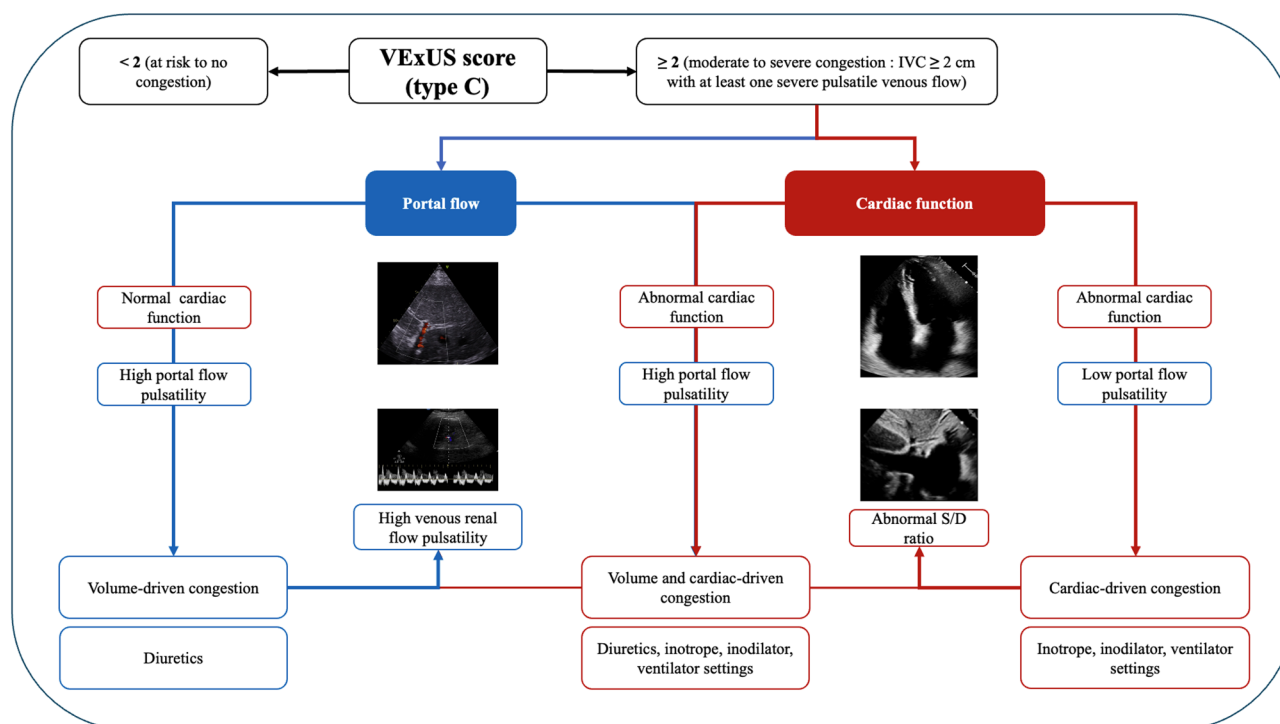


Fig. 1 Interpretation of the VExUS score (type C) in relation to each component. A VExUS score < 2 suggests minimal to no congestion, whereas a VExUS score ≥ 2 indicates moderate to severe congestion. The figure illustrates how portal venous flow and cardiac function interact to differentiate volume-driven versus cardiac-driven congestion. The renal venous flow and hepatic venous flow serve to confirm whether congestion is volume-driven, cardiac-driven or both. Comprehensive cardiac function assessment includes biventricular systolic and diastolic function, along with ventriculo-arterial coupling evaluation. S/D ratio refers to the ratio of S wave to D wave in hepatic vein flow. IVC: inferior vena cava

Abbreviations

IVC Inferior vena cava
ICU Intensive care unit
VExUS Venous Excess Ultrasound

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